REMARKS

Claims 4-21 have been canceled. Claim 1 has been amended. Claims 1-3 are currently pending in this application. Support for the language "beta-amyloid-induced vasoactivity" can be found on page 4, 2nd full paragraph. Support for the language "individuals with Alzheimer's Disease or related disorders" can be found on page 9, 1st and 2nd paragraphs. Support for the language "antagonizing the phospholipase A2/arachidonic acid/5-lipoxygenase/cyclo-oxygenase-2" can be found on page 11, 2nd full paragraph. Support for the language "administering a pharmaceutically effective amount of a PLA_2 inhibitor to the individual" can be found on page 11, 3rd full paragraph. No new matter has been added. In view of these amendments and of the following remarks, Applicants believe that all the asserted rejections are in condition for withdrawal and all the claims are in condition for allowance.

The Examiner is requiring that Applicants submit a new Oath or Declaration. Accordingly, Applicants have submitted a new Oath or Declaration in compliance with 37 C.F.R. 1.67(a) under separate cover.

The Examiner states that if Applicants desire priority under 35 U.S.C. 119(e) based upon a previously filed application, specific reference to the earlier filed application must be made in the instant application. Applicants notice, however, that a Preliminary Amendment, filed January 11, 2001, amended the specification to include the section heading "Cross Reference to Related Applications," in which priority was claimed to U.S. Provisional Application No. 60/092,570, filed July 13, 1998. A copy of the Preliminary Amendment is enclosed herein for the Examiner's review.

Claims 1-3 stand rejected under 35 U.S.C. 112, second paragraph, for purported indefiniteness. The Examiner asserts that claim 1 is essentially incomplete by lacking essential steps needed to perform the claimed method. Accordingly, claim 1 has been amended to recite a method of modifying β-amyloid-induced vasoactivity in individuals with Alzheimer's Disease or related disorders comprised of antagonizing the phospholipase A2/arachidonic acid/5-lipoxygenase/cyclo-oxygenase-2 pro-inflammatory pathway by administering a pharmaceutically effective amount of a phospholipase A2 inhibitor to the individual. Applicants submit that claim 1 as amended now adequately fulfills the requirement to particularly point out and distinctly claim the subject matter of the present invention.

Claims 1 and (2 or 3) stand rejected under 35 U.S.C. 102(b) for purported anticipation by Adams et al., and claims 1 and (2 or 3) stand rejected under 35 U.S.C. 102(a) for purported anticipation by Jackson et al. The Examiner states that Applicants did not identify the claims readable on the elected species. Applicants now identify claim 3 as readable on the elected species. The Examiner asserts that Adams et al. disclose treating cytokine mediated diseases by administering to a mammal in need thereof a p38 MAP kinase inhibitor compound. The Examiner further asserts that Jackson et al. disclose the administration of SB 220025, an inhibitor of p38 MAP kinase orally to mice to study the inhibitor's effect on inflammatory cytokine production or angiogenesis. Finally, the Examiner asserts that modifying vasoactivity by regulating a soluble alpha [sic]-beta pro-inflammatory pathway would be inherent.

The present invention inheres in the discovery that modifying β-amyloid vasoactivity in individuals with Alzheimer's Disease or related disorders by manipulating specific signal transduction pathways via the administration of drugs that antagonize, i.e., block, inhibit, inactivate, the phospholipase A2/arachidonic acid/5-lipoxygenase/cyclo-oxygenase-2 (PLA_2/AA/LOX/COX) pro-inflammatory pathway, may affect the course of Alzheimer's Disease or related disorders in such individuals.

In contrast, Adams et al. disclose compounds and use thereof to inhibit cytokine production and consequent inflammatory reactions in mammals, particularly for the veterinary treatment of viral infections, and Jackson et al. disclose the use of p38 MAP kinase inhibitors to examine the role of cytokine mediation of inflammatory angiogenesis.

Neither Adams et al. or Jackson et al. teach nor suggest that their compounds can modify beta-amyloid-induced vasoactivity by specifically antagonizing the PLA_2/AA/LOX/COX pro-inflammatory pathway in individuals with Alzheimer's Disease or related disorders. Applicants submit that the mere teaching of anti-inflammatory compounds and use thereof does not anticipate the unexpected discovery of the present invention that the administration of drugs capable of antagonizing the PLA_2 signal transduction pathway results in modification of β-amyloid vasoactivity, which thus may affect the course of Alzheimer's Disease or related disorders in individuals afflicted thereof. Applicants further submit that the mere administration of a compound, i.e., a p38 MAP kinase inhibitor, to treat inflammatory reactions or to examine the role of inflammatory mediators, does not teach inherently the new and unexpected finding of

the present invention that drugs that antagonize the PLA_2/AA/LOX/COX pro-inflammatory pathway are capable of modifying β-amyloid vasoactivity in individuals with Alzheimer's Disease or related disorders.

For all the foregoing reasons, claims 1-3 are patentable over the cited prior art and in condition for allowance. Reconsideration of the rejections and allowance of pending claims 1-3 are respectfully requested.

Respectfully submitted,

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